	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	4	glycoprotein same albumen same (chicken adj egg)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/09/30 08:23			0

=> d his

(FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
07:46:57 ON 30 SEP 2002

L1 513834 S GLYCOPROTEIN
L2 85075 S (HELICOBACTER PYLORI) OR (H. PYLORI)
L3 32273 S UREASE
L4 8106 S L2 (P) L3
L5 26 S L1 (P) L4
L6 9 DUPLICATE REMOVE L5 (17 DUPLICATES REMOVED)
L7 6 S L6 (P) BIND?
L8 16186 S WHEY (P) MILK
L9 11988 S ALBUMIN (P) EGG
L10 1 S L6 (P) (L8 OR L9)
L11 0 S L10 NOT L6
L12 0 S L2 (P) INHIBIT? SAME COLONIZ?
L13 635 S L2 (P) INHIBIT? (P) COLONIZ?
L14 16 S L1 (P) L13
L15 5 DUPLICATE REMOVE L14 (11 DUPLICATES REMOVED)
L16 3 S L15 NOT L6
L17 20 S L3 (P) (AFFINITY CHROMATOGRAPHY) (P) IMMOBILIZ?
L18 1 S L17 (P) L1
L19 0 S L18 NOT (L16 OR L6)
L20 37224 S (GASTROINTESTINAL DISEASE)
L21 0 S L20 (P) L15
L22 3733 S INHIBIT (P) (GASTRIC ACID) (P) SECRET?
L23 0 S L15 AND L22

=> log y

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FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002

=> file medline caplus biosis embase scisearch agricola
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 07:46:57 ON 30 SEP 2002

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FILE 'AGRICOLA' ENTERED AT 07:46:57 ON 30 SEP 2002

=> s glycoprotein
L1      513834 GLYCOPROTEIN

=> s (helicobacter pylori) or (H. pylori)
L2       85075 (HELICOBACTER PYLORI) OR (H. PYLORI)

=> s urease
L3       32273 UREASE

=> s l2 (p) l3
L4        8106 L2 (P) L3

=> s l1 (p) l4
L5          26 L1 (P) L4

=> duplicate remove l5
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L5
L6            9 DUPLICATE REMOVE L5 (17 DUPLICATES REMOVED)

=> d l6 1-9 ibib abs

L6   ANSWER 1 OF 9  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:760034  CAPLUS
DOCUMENT NUMBER:          135:278059
TITLE:                    Glycoprotein having inhibitory activity against
                          Helicobacter pylori colonization
INVENTOR(S):              Kodama, Yoshikatsu; Kimura, Nobutake
PATENT ASSIGNEE(S):       Ghen Corporation, Japan; Nisshin Flour Milling Co.,
                          Ltd.
SOURCE:                   Eur. Pat. Appl., 16 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     EP 1145644         A2   20011017       EP 2001-400969   20010413
     EP 1145644         A3   20020612
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
     JP 2001294600      A2   20011023       JP 2000-113913   20000414
```

US 2001044120 A1 20011[illegible] US 2001-833637 20010413
CN 1331250 A 20020[illegible] CN 2001-123320 200104[illegible]
PRIORITY APPLN. INFO.: JP 2000-113913 A 20000414
AB An inhibitor of ***Helicobacter*** ***pylori*** colonization in the stomach comprises as an active ingredient a ***glycoprotein*** which specifically binds to ***H*** . ***pylori*** ***urease*** . This ***glycoprotein*** is isolated and purified from a ***glycoprotein*** -contg. substance, esp. that derived from bovine milk whey or albumen of chicken eggs by affinity chromatog. using a column on which ***H*** . ***pylori*** ***urease*** is immobilized. The ***glycoprotein*** is able to effectively inhibit ***H*** . ***pylori*** colonization, and thus is useful for the prevention or treatment of diseases caused by infection of ***H*** . ***pylori*** such as peptic ulcers. A food and medicament comprising the inhibitor are also provided.

L6 ANSWER 2 OF 9 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000403971 MEDLINE
DOCUMENT NUMBER: 20389972 PubMed ID: 10930371
TITLE: Acid-dependent adherence of Helicobacter pylori urease to diverse polysaccharides.
AUTHOR: Icatlo F C; Goshima H; Kimura N; Kodama Y
CORPORATE SOURCE: Immunology Research Institute, Ghen Corp., Sano, Gifu City, Japan.. irig@ghen.co.jp
SOURCE: GASTROENTEROLOGY, (2000 Aug) 119 (2) 358-67.
Journal code: 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000901
Last Updated on STN: 20000901
Entered Medline: 20000822
AB BACKGROUND & AIMS: The significance of acid-primed recognition of ligands by ***Helicobacter*** ***pylori*** ***urease*** is unknown. This study aimed to further characterize the specificity of ***urease*** adherence in vitro and verify whether specific inhibition will translate into in vivo suppression of colonization. METHODS: A highly sensitive competitive enzyme-linked ligand capture assay was used to quantify the capacity of each test inhibitor to compete with labeled mucin for binding sites on immobilized native ***urease*** . A model polymer that strongly bound ***urease*** was used in an in vivo trial using euthymic hairless mice as an infection model. RESULTS: The blockage of ***urease*** -gastric mucin interaction by certain inhibitors revealed an acid-functional lectin-like activity by ***urease*** , specifically recognizing bacterial lipopolysaccharides and certain species of polysaccharides, nonbacterial glycolipids, and ***glycoproteins*** . Dextran sulfate significantly ($P < 0.01$) suppressed colonization of mice by ***H*** . ***pylori*** when given before and/or after challenge. CONCLUSIONS: The acid-driven high-affinity adherence of ***H*** . ***pylori*** ***urease*** to mucin and lipopolysaccharides contributes to gastric mucosal colonization by the bacterium based on in vivo targeting experiments using specific polysaccharides in a mouse model with acute infection. Acid-functional ***urease*** -homing polysaccharides that can interfere with ***urease*** -mucin or ***H*** . ***pylori*** whole cell-mucin interaction in vitro can significantly interfere with colonization by the bacterium in vivo.

L6 ANSWER 3 OF 9 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1999:863987 SCISEARCH
THE GENUINE ARTICLE: 253AT
TITLE: Live attenuated Salmonella: a paradigm of mucosal vaccines
AUTHOR: Sirard J C (Reprint); Niedergang F; Kraehenbuhl J P
CORPORATE SOURCE: UNIV LAUSANNE, SWISS INST EXPT CANC RES, CH-1066 EPALINGES, SWITZERLAND (Reprint); UNIV LAUSANNE, INST BIOCHEM, CH-1066 EPALINGES, SWITZERLAND
COUNTRY OF AUTHOR: SWITZERLAND
SOURCE: IMMUNOLOGICAL REVIEWS, (OCT 1999) Vol. 171, pp. 5-26.
Publisher: MUNKSGAARD INT PUBL LTD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.
ISSN: 0105-2896.

DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 211
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Two key steps control immune responses in mucosal tissues: the sampling and transepithelial transport of antigens, and their targeting into professional antigen-presenting cells in mucosa-associated lymphoid tissue. Live Salmonella bacteria use strategies that allow them to cross the epithelial barrier of the gut, to survive in antigen-presenting cells where bacterial antigens are processed and presented to the immune cells, and to express adjuvant activity that prevents induction of oral tolerance. Two Salmonella serovars have been used as vaccines or vectors, S. typhimurium in mice and S. typhi in humans. S. typhimurium causes gastroenteritis in a broad host range, including humans, while S. typhi infection is restricted to humans. Attenuated S. typhimurium has been used successfully in mice to induce systemic and mucosal responses against more than 60 heterologous antigens. This review aims to revisit S. typhimurium-based vaccination, as an alternative to S. typhi, with special emphasis on the molecular pathogenesis of S. typhimurium and the host response. We then discuss how such knowledge constitutes the basis for the rational design of novel live mucosal vaccines.

L6 ANSWER 4 OF 9 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97162351 MEDLINE
DOCUMENT NUMBER: 97162351 PubMed ID: 9009338
TITLE: Inhibition of Helicobacter pylori binding to gastrointestinal epithelial cells by sialic acid-containing oligosaccharides.
AUTHOR: Simon P M; Goode P L; Mobasseri A; Zopf D
CORPORATE SOURCE: Neose Technologies, Inc., Horsham, Pennsylvania 19044, USA.. SimonPM@AOL.com
SOURCE: INFECTION AND IMMUNITY, (1997 Feb) 65 (2) 750-7.
Journal code: 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970306
Last Updated on STN: 19970306
Entered Medline: 19970221

AB Helicobacterpylori, the ulcer pathogen residing in the human stomach, binds to epithelial cells of the gastric antrum. We have examined binding of 13 bacterial isolates to epithelial cell lines by use of a sensitive microtiter plate method in which measurement of bacterial ***urease*** activity provides the means for quantitation of bound organisms. Several established human gastrointestinal carcinoma cell lines grown as monolayers were compared for suitability in these assays, and the duodenum-derived cell line HuTu-80 was selected for testing bacterial binding inhibitors. When bacteria are pretreated with oligosaccharides, ***glycoproteins*** , and glycolipids, a complex picture of bacterial-epithelial adherence specificities emerges. Among the monovalent inhibitors tested, 3'-sialyllactose (NeuAc alpha2-3Gal beta1-4Glc; 3'SL) was the most active oligosaccharide, inhibiting adherence for recent clinical isolates of ***H*** . ***pylori*** with a millimolar 50% inhibitory concentration (IC50). Its alpha2-6 isomer (6'SL) was less active. Most of the recent clinical isolates examined were inhibited by sialyllactose, whereas long-passaged isolates were insensitive. Among the long-passaged bacterial strains whose binding was not inhibited by 3'SL was the strain ATCC 43504, also known as NCTC 11637 and CCUG 17874, in which the proposed sialyllactose adhesin was recently reported to lack surface expression (P. G. O'Toole, L. Janzon, P. Doig, J. Huang, M. Kostrzynska, and T. H. Trust, J. Bacteriol. 177:6049-6057, 1995). Pretreatment of the epithelial monolayer with neuraminidase reduced the extent of binding by those bacteria that are sensitive to inhibition by 3'SL. Other potent inhibitors of bacterial binding are the ***glycoproteins*** alpha1-acid ***glycoprotein*** , fetuin, porcine gastric and bovine submaxillary mucins, and the glycolipid sulfatide, all of which present multivalent sialylated and/or sulfated galactosyl residues under the conditions of the binding assay. Consistent with this pattern, a multivalent neoglycoconjugate containing 20 mol of 3'SL per mol

of human serum albumin inhibited bacterial binding with micromolar IC50. The ***H*** . ***pylori*** isolate most sensitive to inhibition by 3'SL was least sensitive to inhibition by sulfatide, gastric mucin, and other sulfated oligosaccharides. Bacteria that have been allowed to bind epithelial cells are also effectively detached by 3'SL. These results describe a heterogeneous adherence repertoire for these bacteria, but they also confirm the critical role of the 3'SL structure on human gastric epithelial cells as an adherence ligand for recent isolates of ***H*** . ***pylori*** .

L6 ANSWER 5 OF 9 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97256726 MEDLINE
DOCUMENT NUMBER: 97256726 PubMed ID: 9099625
TITLE: Sulfatides inhibit binding of Helicobacter pylori to the gastric cancer Kato III cell line.
AUTHOR: Wadstrom T; Hirmo S; Novak H; Guzman A; Ringner-Pantzar M; Utt M; Aleljung P
CORPORATE SOURCE: Department of Medical Microbiology, University of Lund, Solvegatan 23, Lund S-22362, Sweden.
SOURCE: CURRENT MICROBIOLOGY, (1997 May) 34 (5) 267-72.
Journal code: 7808448. ISSN: 0343-8651.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Biotechnology
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970612
Last Updated on STN: 19990129
Entered Medline: 19970602
AB ***Helicobacter*** ***pylori*** adhere to Kato III and Hela S3 cells in monolayer cultures. To explore whether cell surface glycoconjugates on these two cell lines mediate binding of ***H*** . ***pylori*** , various carbohydrates, ***glycoproteins*** , and glycolipids were tested to inhibit ***H*** . ***pylori*** cell adhesion. The adhesion was measured (i) with a ***urease*** -based assay and (ii) by cells stained with fluorescein. Sodium periodate and sialidase treatment (but not alpha- or beta-galactosidase, heparitinase,lysozyme, or trypsin) inhibited ***H*** . ***pylori*** binding to both cell lines.Sulfatides and sulfated glycoconjugates (50 microg/ml) but not heparin or a number of simple carbohydrates inhibited binding (1 mg/ml). The two ***H*** . ***pylori*** strains studied (CCUG 17874 and strain 25) showed high binding of soluble 125I-labeled heparin and other sulfated carbohydrate compounds.

L6 ANSWER 6 OF 9 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 92165602 EMBASE
DOCUMENT NUMBER: 1992165602
TITLE: Antibodies against Helicobacter pylori inhibit the adhesion of this organism to the gastric mucosal surface.
AUTHOR: Tanaka N.; Kuwayama H.; Sunairi M.; Nakajima M.
CORPORATE SOURCE: Dept. of Internal Medicine (III), School of Medicine, Nihon University, 1-7-3 Kandasurugadai,Chiyoda-ku, Tokyo 101, Japan
SOURCE: European Journal of Gastroenterology and Hepatology, (1992) 4/SUPPL. 1 (S67-S69).
ISSN: 0954-691X CODEN: EJGHES
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 004 Microbiology
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Objectives. The adhesion properties of a strain of ***Helicobacter*** ***pylori*** were studied in an in vitro system. Design. ***H*** . ***pylori*** adhesion to the gastric mucosal surface was examined in an in vitro system, using polystyrene assay plates coated with gastric mucus derived from various sources. The adhesion was further studied using fractions from mucin, glycosylated beads and anti- ***H*** . ***pylori*** antibodies. Method. The numbers of ***H*** . ***pylori*** cells adhering to the plates were estimated by measuring ***urease*** activity. Results. There was strong adhesion to partly purified mucin from a porcine stomach, but only weak adhesion to mucus

derived from cattle. ***H*** . ***pylori*** adhered to galactosylated beads, acidic ***glycoprotein*** and the g[illegible]olipid components of porcine mucin. Bacterial adhesion was inhibited not only by whole molecules but also by the antigen-binding fragment of anti- ***H*** . ***pylori*** immunoglobulin G. Conclusions. ***H*** . ***pylori*** appeared to have an affinity for galactosylated beads. Further, we suggest that the use of antibodies might be helpful in treating the ***H*** . ***pylori*** infection.

L6 ANSWER 7 OF 9 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 92003478 MEDLINE
DOCUMENT NUMBER: 92003478 PubMed ID: 1912416
TITLE: Virulence and pathogenicity of Helicobacter pylori.
AUTHOR: Marshall B J
CORPORATE SOURCE: Department of Internal Medicine, University of Virginia Health Sciences Center, Charlottesville 22908.
SOURCE: JOURNAL OF GASTROENTEROLOGY AND HEPATOLOGY, (1991 Mar-Apr) 6 (2) 121-4. Ref: 28
Journal code: 8607909. ISSN: 0815-9319.
PUB. COUNTRY: Australia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199111
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911108
AB ***H*** . ***pylori*** is a highly virulent organism as evidenced by its low infective dose and widespread high prevalence in human populations. Its virulence is achieved through its ability to survive in a moist environment and its massive ***urease*** production which allows it to survive in the acidic gastric juice long enough to colonize the gastric mucus. Gastric colonization is facilitated by cell wall associated lectins which permit the bacterium to bind to gastric mucus and the gastric epithelial cell. Once in this location, ***H*** . ***pylori*** produces several enzymes which may harm the gastric epithelium, particularly ***urease*** (through ammonia generation) and phospholipases A and C. ***H*** . ***pylori*** also weakens the gastric mucous layer by digesting its ***glycoproteins*** and lipids, making the mucus less hydrophobic and more water soluble. ***Helicobacter*** ***pylori*** attracts phagocytic cells, inducing both acute and chronic inflammation as well as an antibody response. Persistence of ***H*** . ***pylori*** in the mucosa may be enhanced by its cytotoxin and catalase production, by which it survives after phagocytosis by neutrophils.

L6 ANSWER 8 OF 9 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 91147586 MEDLINE
DOCUMENT NUMBER: 91147586 PubMed ID: 1997534
TITLE: Breakdown of gastric mucus in presence of Helicobacter pylori.
AUTHOR: Sidebotham R L; Batten J J; Karim Q N; Spencer J; Baron J H
CORPORATE SOURCE: Department of Surgery, Royal Postgraduate Medical School, Hammersmith Hospital, London.
SOURCE: JOURNAL OF CLINICAL PATHOLOGY, (1991 Jan) 44 (1) 52-7.
Journal code: 0376601. ISSN: 0021-9746.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910419
Last Updated on STN: 19910419
Entered Medline: 19910404
AB The potential of ***Helicobacter*** ***pylori*** to degrade gastric mucus was examined. Colonies of ***H*** ***pylori*** cultured from antral mucosal biopsy specimens of patients with non-autoimmune gastritis were washed with sterile saline, passed through a sterilisation filter, and the filtrate examined for ***urease*** , protease, and mucolytic activity. The filtrate failed to hydrolyse bovine

serum albumin, or to degrade [redacted]able mucus ***glycoprotein*** structures of high particle w[redacted]ght that had been separated fr[redacted]human gastric mucus on Sepharose 2B. The high particle weight mucus ***glycoprotein*** was, however, extensively degraded when incubated with ***H*** ***pylori*** filtrate (which possessed ***urease*** activity) in the presence of 2 M urea, to release fragments of Mr approximately 2 X 10(6). The high particle weight mucus ***glycoprotein*** was also broken down to a comparable extent when incubated with Jack bean ***urease*** in the presence of 2 M urea, or 1 M ammonium carbonate, or 40 mM carbonate-bicarbonate buffer (pH 8.7), but not when treated with 4 M urea alone, or Jack bean ***urease*** alone. These results indicate that the loss of high particle weight mucus ***glycoprotein*** in gastric mucus from patients with gastritis and gastric ulcers is unlikely to be due to the mucolytic action of an extra-cellular protease produced by ***H*** ***pylori*** , but it may result from the destabilising effects of a carbonate-bicarbonate buffer, generated at the mucosal surface when ***H*** ***pylori*** ***urease*** hydrolyses transuded plasma urea.

L6 ANSWER 9 OF 9 MEDLINE
ACCESSION NUMBER: 90306646 MEDLINE
DOCUMENT NUMBER: 90306646 PubMed ID: 2194877
TITLE: [Does Helicobacter pylori have a direct proteolytic effect in ulcerative disease?].
L'Helicobacter pylori esercita un'azione proteolitica diretta in corso di malattia ulcerosa?.
AUTHOR: Tessaro P; Di Mario F; Vianello F; Dal Santo P; Germana B; Plebani M; Faggian D; Del Favero G; Naccarato R
CORPORATE SOURCE: Cattedra e Divisione di Gastroenterologia, Universita di Padova.
SOURCE: GIORNALE DI CLINICA MEDICA, (1990 Mar) 71 (3) 173-8, 181.
Journal code: 0413411. ISSN: 0017-0275.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199008
ENTRY DATE: Entered STN: 19900921
Last Updated on STN: 19900921
Entered Medline: 19900816

AB ***Helicobacter*** ***pylori*** (H.p.), has been shown, experimentally, to exert a proteolytic activity against mucous fractions. Aim of this study was to assess the prevalence of H.p. in peptic ulcer and to analyze its possible influence on gastric mucus components, on peptic activity in gastric juice and the possible action on peptic secretion. 223 patients undergoing upper gastrointestinal endoscopy were analyzed for the presence of H.p. in the mucosa: 99 duodenal ulcer patients (D.U.), 58 gastric ulcer patients (D.U.) and 66 dyspeptic subjects. In each patients, three contiguous gastric biopsies were taken at the antrum: the first was evaluated for gastritis (Whitehead Criteria), the two other analyzed for H.p. with a rapid ***urease*** test. In a subgroup of 25 D.U. and 18 G.U. patients, two other biopsies were taken at the fundus corpus of the stomach, to evaluate peptic secretion. To determinate mucous components (acid and neutral ***glycoproteins*** , galactose and N-acetylneuraminic acid), gastric juice samples were collected during endoscopy. H.p. was present in 89% of antral biopsies in D.U., in 56% of G.U. and in 51% of D., and was associated to antral gastritis. As regard gastric juice components, we observed an increase and a decrease of acid ***glycoproteins*** , respectively, in D.U. and G.U. patients with H.p. infection. An increase of peptic activity has been found in the gastric juice of both gastric and duodenal ulcer patients H.p. positive (G.U. p less than 0.05). On the contrary, no significant differences were observed on peptic activity in the fundus-corpus biopsies between H.p. positive and H.p. negative patients.(ABSTRACT TRUNCATED AT 250 WORDS)

=> d his

(FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 07:46:57 ON 30 SEP 2002

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L1       513834 S GLYCOPROTEIN
L2        85075 S (HELICOBACTER PYLORI) OR (H. PYLORI)
L3        32273 S UREASE
L4         8106 S L2 (P) L3
L5           26 S L1 (P) L4
L6            9 DUPLICATE REMOVE L5 (17 DUPLICATES REMOVED)

=> s l6 (p) bind?
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L41 (P) BIND?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L47 (P) BIND?'
L7            6 L6 (P) BIND?

=> s whey (p) milk
L8       16186 WHEY (P) MILK

=> s albumin (p) egg
L9       11988 ALBUMIN (P) EGG

=> s l6 (p) (l8 or l9)
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L68 (P) '
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L74 (P) '
L10           1 L6 (P) (L8 OR L9)

=> s l10 not l6
L11           0 L10 NOT L6

=> d his

     (FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     07:46:57 ON 30 SEP 2002
L1       513834 S GLYCOPROTEIN
L2        85075 S (HELICOBACTER PYLORI) OR (H. PYLORI)
L3        32273 S UREASE
L4         8106 S L2 (P) L3
L5           26 S L1 (P) L4
L6            9 DUPLICATE REMOVE L5 (17 DUPLICATES REMOVED)
L7            6 S L6 (P) BIND?
L8        16186 S WHEY (P) MILK
L9        11988 S ALBUMIN (P) EGG
L10           1 S L6 (P) (L8 OR L9)
L11           0 S L10 NOT L6

=> s l2 (p) inhibit? same coloniz?
L12           0 L2 (P) INHIBIT? SAME COLONIZ?

=> s l2 (p) inhibit? (p) coloniz?
L13         635 L2 (P) INHIBIT? (P) COLONIZ?

=> s l1 (p) l13
L14          16 L1 (P) L13

=> duplicate remove l14
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L14
L15           5 DUPLICATE REMOVE L14 (11 DUPLICATES REMOVED)

=> s l15 not l6
L16           3 L15 NOT L6

=> d l16 1-3 ibib abs

L16  ANSWER 1 OF 3     MEDLINE
ACCESSION NUMBER:    2000158106     MEDLINE
DOCUMENT NUMBER:     20158106   PubMed ID: 10695559
TITLE:               Helicobacter pylori lipopolysaccharide-mediated gastric and
```

extragastric pathology.
AUTHOR: Moran A P
CORPORATE SOURCE: Department of Microbiology, National University of Ireland. Galway.. anthony.moran@nuigalway.ie
SOURCE: JOURNAL OF PHYSIOLOGY AND PHARMACOLOGY, (1999 Dec) 50 (5) 787-805. Ref: 97
Journal code: 9114501. ISSN: 0867-5910.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000407
Last Updated on STN: 20000407
Entered Medline: 20000328

AB Lipopolysaccharides (LPS) are a family of toxic phosphorylated glycolipids in the outer membrane of Gram-negative bacteria, including ***Helicobacter*** ***pylori*** , and are composed of a lipid moiety (termed lipid A), a core oligosaccharide, and a polymeric O-specific polysaccharide chain. Compared with LPS of other bacteria, ***H*** . ***pylori*** LPS and lipid A induce low immunological activities in a range of test systems. Nevertheless, these reduced levels of LPS-induced cytokines and toxic oxygen radicals can contribute, with those induced by bacterial proteins, to the ***H*** . ***pylori*** -associated inflammatory response. Whether the ability of ***H*** . ***pylori*** LPS to induce low production of both procoagulant activity and plasminogen activator ***inhibitor*** type 2 by human mononuclear cells contributes to localized inflammatory responses alone and, in addition, play a role in extragastric pathology remains an open question. The core oligosaccharide of ***H*** . ***pylori*** LPS, in part with a 25 kDa protein adhesin, mediates the binding of the bacterium to the host ***glycoprotein*** laminin, and hence interferes with gastric cell receptor-laminin interaction in the basement membrane. Also affecting mucosal integrity, the core sugars of certain ***H*** . ***pylori*** strains, particularly those associated with gastric ulceration, have been implicated in pepsinogen induction, but this is a strain-dependent phenomenon. Of particular interest, the O-chains of a large proportion of ***H*** . ***pylori*** strains mimic Lewis (Le) antigens. Although investigations have focussed on the role of these antigens in ***H*** . ***pylori*** -associated autoimmunity, which remains to be unequivocally established, other pathogenic consequences of Lewis mimicry are becoming apparent. Expression of Lewis antigens may be crucial for ***H*** . ***pylori*** ***colonization*** and adherence and, by aiding bacterial interaction with the gastric mucosa, thereby aid delivery of secreted products, and hence influence the inflammatory response.

L16 ANSWER 2 OF 3 MEDLINE
ACCESSION NUMBER: 92391434 MEDLINE
DOCUMENT NUMBER: 92391434 PubMed ID: 1381553
TITLE: Glycosulfatase activity of H. pylori toward human gastric mucin: effect of sucralfate.
AUTHOR: Slomiany B L; Murty V L; Piotrowski J; Grabska M; Slomiany A
CORPORATE SOURCE: Research Center, New Jersey Dental School, University of Medicine and Dentistry of New Jersey, Newark.
CONTRACT NUMBER: AA05858-11 (NIAAA)
DK31684-15 (NIDDK)
SOURCE: AMERICAN JOURNAL OF GASTROENTEROLOGY, (1992 Sep) 87 (9) 1132-7.
Journal code: 0421030. ISSN: 0002-9270.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199210
ENTRY DATE: Entered STN: 19921023
Last Updated on STN: 19960129
Entered Medline: 19921008

AB ***Colonization*** of gastric mucosa by ***Helicobacter*** ***pylori*** , a bacterium implicated in the etiology of gastric disease,

involves the cell surface sulfated glycosphingolipid receptors for the attachment. Evidence has also been obtained recently that sulfated mucus ***glycoproteins*** have the ability to interfere with this process. Here, we show that ***H*** . ***pylori*** displays glycosulfatase activity, and report the specificity of this enzyme toward gastric mucosal sulfated ***glycoproteins*** and glycolipids. With 35S-labeled human gastric sulfated mucin as substrate, the enzyme activity was identified in the extracellular material elaborated by the bacterium. The glycosulfatase exhibited maximum activity at pH 5.7 in the presence of Triton X-100 and CaCl2, and gave on SDS-PAGE a protein band of 30 kDa. Specificity studies revealed that the enzyme effectively caused desulfation of N-acetylglucosamine-6-sulfate and galactose-6-sulfate present in carbohydrate chains of gastric mucins, as well as that of glucose-6-sulfate, a constituent of mucus glyceroglucolipids. However, the ***H*** . ***pylori*** glycosulfatase was ineffective toward galactosyl- and lactosylceramide sulfates which serve as receptors for this bacterium attachment and contain the sulfate ester group at C-3 of galactose. The glycosulfatase activity toward human sulfated gastric mucin was ***inhibited*** by sucralfate. The ***inhibitory*** effect was proportional to the concentration of sucralfate up to 120 micrograms/ml, at which a 78% decrease in mucin desulfation occurred. The results demonstrate that ***H*** . ***pylori*** , through its glycosulfatase activity, affects the sulfated mucin and glyceroglucolipid content of the protective mucus layer, and that antiucler drug sucralfate is able to counteract the detrimental action of this enzyme.

L16 ANSWER 3 OF 3 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000197399 EMBASE
TITLE: Role of oligosaccharides and glycoconjugates in intestinal host defense.
AUTHOR: Dai D.; Nanthkumar N.N.; Newburg D.S.; Walker W.A.
CORPORATE SOURCE: Dr. W.A. Walker, Division of Nutrition, Harvard Medical School, C. Hosp./Massachusetts General Hosp., 300 Longwood Avenue, Boston, MA 02115, United States
SOURCE: Journal of Pediatric Gastroenterology and Nutrition, (2000) 30/SUPPL. 2 (S23-S33).
Refs: 96
ISSN: 0277-2116 CODEN: JPGND6
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 004 Microbiology
007 Pediatrics and Pediatric Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The attachment of microbes to carbohydrates moieties on the host cell surface is considered essential for successful ***colonization*** and infection. Adding sugars to MVM membrane proteins and lipids (glycosylation) is an important host determinant in microbial ***colonization*** of the intestine. The enzymes responsible for glycosylation are glycosyltransferases. Our studies and others have shown that glycosyltransferases and microbial receptors are under developmental regulation. Intrinsic genetic control, hormones (glucocorticoids, insulin and thyroxine), and external factors (diet and bacterial ***colonization***) may affect the ontogeny of these enzymes and the expression of microbial receptors. Therefore, the developmental control of microbial receptors in the gastrointestinal tract may in part contribute to the altered host sensitivity to intestinal infection in infancy. Probiotics and some trophic factors may also protect the gastrointestinal tract through differential glycosylation. In the future, it may also be possible to ***inhibit*** microbial attachment by blocking with oligosaccharides or glycoconjugates specific for the appropriate lectins. The molecular nature of microbial receptors in intestinal epithelial cells underscores the importance of intestinal surface carbohydrate expression in host-microbe interaction. With improved techniques for characterizing receptor binding and the receptor's structure - i.e., the availability of several human intestinal models (organ culture of human fetus intestine, primary culture of human fetus intestinal epithelial cells, the H-4 cell line, and Caco-2 cell line) and of carbohydrate-specific monoclonal antibodies, we may identify additional membrane receptors and the receptor sugar sequences in the near future. We may isolate glycoconjugates from human intestinal tissue, then identify them structurally using mass spectrometry and nuclear magnetic resonance spectroscopy. We may test the

binding of microbial ligands [redacted] epithelial surfaces with ***glycoproteins*** or gl[redacted]olipids. Subsequent studies on [redacted]e intestinal expression and developmental regulation of individual glycosyltransferases can then be pursued. Recently, a transgenic mouse model has been used to study ***Helicobacter*** ***pylori*** infection, in which the receptor, the primate-specific Lewisb (95), was expressed in the mouse gastrointestinal tract by transfection with a human .alpha.-1,3/4-fucosyltransferase (96). In the future, the potential transgenic animal models by transfection with constructs for specific glycosyl-transferase(s) will be used to examine the role of oligosaccharides and glycoconjugates in regulating cellular differentiation and the host-microbe interaction. In the same manner, the use of molecular and cell biologic techniques in intestinal cell lines and in primary cultures of human enterocytes or organ culture of human fetal, neonatal, and adult intestine will help to determine the relationship between developmental regulation of intestinal microbial receptors and postreceptor-effector events. By understanding the molecular nature of microbial receptors and their effector responses in the intestine, the developmental programming and environmental influence on receptor expression, and the effector response and the biologic significance in neonatal host defenses, new approaches may soon be available in the prevention and treatment of infants with infectious intestinal diseases of various origins.

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=> d his

     (FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     07:46:57 ON 30 SEP 2002
L1       513834 S GLYCOPROTEIN
L2        85075 S (HELICOBACTER PYLORI) OR (H. PYLORI)
L3        32273 S UREASE
L4         8106 S L2 (P) L3
L5           26 S L1 (P) L4
L6            9 DUPLICATE REMOVE L5 (17 DUPLICATES REMOVED)
L7            6 S L6 (P) BIND?
L8        16186 S WHEY (P) MILK
L9        11988 S ALBUMIN (P) EGG
L10           1 S L6 (P) (L8 OR L9)
L11           0 S L10 NOT L6
L12           0 S L2 (P) INHIBIT? SAME COLONIZ?
L13         635 S L2 (P) INHIBIT? (P) COLONIZ?
L14          16 S L1 (P) L13
L15           5 DUPLICATE REMOVE L14 (11 DUPLICATES REMOVED)
L16           3 S L15 NOT L6

=> s l3 (p) (affinity chromatography) (p) immobiliz?
L17          20 L3 (P) (AFFINITY CHROMATOGRAPHY) (P) IMMOBILIZ?

=> s l17 (p) l1
L18           1 L17 (P) L1

=> s l18 not (l16 or l6)
L19           0 L18 NOT (L16 OR L6)

=> s (gastrointestinal disease)
L20       37224 (GASTROINTESTINAL DISEASE)

=> s l20 (p) l15
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L160 (P) L169'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L162 (P) L173'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L163 (P) L175'
L21           0 L20 (P) L15

=> d his

     (FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002)
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     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     07:46:57 ON 30 SEP 2002
L1        513834 S GLYCOPROTEIN
L2         85075 S (HELICOBACTER PYLORI) OR (H. PYLORI)
L3         32273 S UREASE
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L5            26 S L1 (P) L4
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L12            0 S L2 (P) INHIBIT? SAME COLONIZ?
L13          635 S L2 (P) INHIBIT? (P) COLONIZ?
L14           16 S L1 (P) L13
L15            5 DUPLICATE REMOVE L14 (11 DUPLICATES REMOVED)
L16            3 S L15 NOT L6
L17           20 S L3 (P) (AFFINITY CHROMATOGRAPHY) (P) IMMOBILIZ?
L18            1 S L17 (P) L1
L19            0 S L18 NOT (L16 OR L6)
L20        37224 S (GASTROINTESTINAL DISEASE)
L21            0 S L20 (P) L15

=> s inhibit (p) (gastric acid) (p) secret?
L22        3733 INHIBIT (P) (GASTRIC ACID) (P) SECRET?

=> s l15 and l22
L23            0 L15 AND L22

=> d his

     (FILE 'HOME' ENTERED AT 07:46:29 ON 30 SEP 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     07:46:57 ON 30 SEP 2002
L1        513834 S GLYCOPROTEIN
L2         85075 S (HELICOBACTER PYLORI) OR (H. PYLORI)
L3         32273 S UREASE
L4          8106 S L2 (P) L3
L5            26 S L1 (P) L4
L6             9 DUPLICATE REMOVE L5 (17 DUPLICATES REMOVED)
L7             6 S L6 (P) BIND?
L8         16186 S WHEY (P) MILK
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L10            1 S L6 (P) (L8 OR L9)
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L12            0 S L2 (P) INHIBIT? SAME COLONIZ?
L13          635 S L2 (P) INHIBIT? (P) COLONIZ?
L14           16 S L1 (P) L13
L15            5 DUPLICATE REMOVE L14 (11 DUPLICATES REMOVED)
L16            3 S L15 NOT L6
L17           20 S L3 (P) (AFFINITY CHROMATOGRAPHY) (P) IMMOBILIZ?
L18            1 S L17 (P) L1
L19            0 S L18 NOT (L16 OR L6)
L20        37224 S (GASTROINTESTINAL DISEASE)
L21            0 S L20 (P) L15
L22         3733 S INHIBIT (P) (GASTRIC ACID) (P) SECRET?
L23            0 S L15 AND L22

=> log y
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
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FULL ESTIMATED COST                                70.67      70.88

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
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